Nasal Obstruction Considerations in Sleep Apnea

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KEYWORDS

• Nasal obstruction • Obstructive sleep apnea • Sleep-disordered breathing
• Nasal surgery • Septoplasty • Turbinate reduction

KEY POINTS

- The nasal airway plays a significant role in breathing during sleep and, therefore, nasal obstruction can result in sleep-disordered breathing.
- Topical nasal steroids can improve sleep quality, but the evidence for their role in the treatment of obstructive sleep apnea (OSA) is not as strong.
- Surgery to correct nasal obstruction has similarly been shown to improve sleep quality but not necessarily sleep apnea.
- Nasal obstruction surgery may facilitate treatment of patients with OSA by improving tolerance and compliance with continuous positive airway pressure.

INTRODUCTION

Obstructive sleep apnea (OSA), characterized by recurrent episodes of upper airway obstructing occurring during sleep, is a very prevalent condition.[1] According to the Wisconsin Sleep Cohort Study, published in 1993, an estimated 9% of women and 24% of men aged 30 to 60 years have this condition in the United States alone.[2] Given the association between weight gain and obesity and OSA,[3] these data were recently reexamined in the context of the global obesity epidemic, and prevalence estimates increased to 17% and 34%, respectively.[4]

Left untreated, the condition is associated with an increased risk for cardiovascular disease. In addition to representing a significant risk factor for hypertension, coronary artery disease, and stroke,[5–7] OSA is associated with an increased risk of diabetes and cancer.[8,9] It also has significant psychosocial implications, impacting cognitive

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function, social interactions, and quality of life (QOL).[10] For these reasons, OSA has emerged to become a significant public health challenge globally.

The first-line treatment of OSA is continuous positive airway pressure (CPAP), which has been shown to reduce the risks of the aforementioned complications, improve QOL, and lower the rates of motor vehicle accidents.[11] Variable compliance to therapy, however, limits its overall effectiveness; 46% to 83% of patients are nonadherent.[12] The mask interface, discomfort from the air pressure required, nasal obstruction and dryness, and psychological and social factors lead to poor acceptance and nonadherence.[13] Although other nonsurgical treatment options exist, such as oral appliances, surgery may play a role in the treatment of this condition.

The severity of sleep apnea is usually assessed by the apnea-hypopnea index (AHI), which is defined as the average number of complete (apnea) and incomplete (hypopnea) obstructive events in 1 hour of sleep. In general, OSA is defined as an AHI of 5 or greater. Five to 14 is defined as mild disease, 15 to 29 as moderate disease, and 30 or greater as severe disease.[14] In general, a 50% reduction in AHI and a final AHI of 20 per hour or less is defined a surgical cure.[15]

NASAL OBSTRUCTION AND SLEEP APNEA

Nasal obstruction is a known risk factor for sleep-disordered breathing secondary to changes in airflow velocity and resistance.[16] As the nose represents the first portal of air entry, nasal pathologic conditions in the form of septal deviation, nasal polyps, turbinate hypertrophy, and rhinitis can contribute to OSA.[17] Moreover, the nose accounts for 50% of the total upper airway resistance; serves many important physiologic functions, including humidification and filtration; and is the preferred breathing route during sleep.[18] In fact, the oral fraction of inhaled ventilation during sleep in healthy subjects with normal nasal resistance is only approximately 4%.[19]

Many pathophysiologic mechanisms have been described to explain the effect on nasal obstruction on sleep-disordered breathing. According to the Starling resistor model, apnea can occur when nasal obstruction generates enough negative intraluminal pressure downstream to cause the compliant soft tissues of the oropharynx to collapse.[20] In the face of significant nasal resistance, a compensatory switch to mouth breathing may occur. Oral breathing in sleep is physiologically unfavorable and unstable, however, and is associated with up to 2.5 times higher resistance secondary to narrowing of the pharyngeal lumen and further posterior collapse of the tongue, resulting in more frequent apneic events.[21] In addition, bypassing the nasal airway leads to less activation of nasal receptors and the nasal-ventilatory reflex, resulting in decreased muscle tone and ventilation secondary to decreased activation of nasal receptors.[22] Finally, significant nitric oxide production occurs in the nose. With mouth breathing, the decreased nitric oxide production leads to changes in the maintenance of muscle tone and changes in spontaneous ventilation and sleep patterns.[23]

There exists a body of clinical and experimental evidence that demonstrates an association between reducing nasal patency and sleep-disordered breathing. Patients with nasal packing experience decreased sleep quality and an increase in frequency of apneic episodes.[24] This finding was demonstrated in patients who undergo nasal packing for epistaxis[25] as well as in patients with packing after septoplasty.[26] Turhan and colleagues[27] specifically demonstrated significantly higher AHI scores and oxygen desaturations in patients treated with nasal packing compared with transseptal suture after septoplasty. Similar to artificially induced nasal obstruction, patients with chronic nasal congestion report sleep-disordered breathing.[28] Several studies have also demonstrated an association between seasonal allergic rhinitis and